Osteoporosis in Men Treated With Androgen Suppression Therapy for Prostate Cancer

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Androgen suppression therapy through the injection of luteinizing hormone-releasing hormone (LHRH) agonist agents has been the mainstay of therapy for patients with advanced or metastatic prostate cancer. Its use has expanded to include patients with prostate-specific antigen recurrence, also known as biochemical relapse (Moul, 1998; Zietman, 1996). The Agency for Health Care Policy and Research (1999) estimated that total 1994 Medicare expenditure for treatment of prostate cancer was almost $1.5 billion, and almost one-third of this was spent on androgen suppression therapy using LHRH agonists. Administration of LHRH agonists, such as goserelin and leuprolide, was the 13th largest category of physician reimbursement by Medicare (Agency for Health Care Policy and Research). In the United States, about 117,000 men with advanced prostate cancer were treated with LHRH agonist therapy in 1996; as the prevalence of advanced prostate cancer rises, its use and subsequent financial costs also are expected to increase (Landis, Murray, Bolden, & Wingo, 1998). Furthermore, men with biochemical relapse who generally are younger and asymptomatic now are faced with long-term use of LHRH agonist therapy. Although patients may tolerate immediate side effects, such as hot flushes, impotence, and loss of libido, healthcare professionals must address new challenges and issues surrounding long-term sequelae of LHRH agonist therapy. The effects of LHRH agonist therapy on bone metabolism, the risk of osteoporosis among men with prostate cancer, and strategies to promote quality of life for these men must be studied.

Men with advanced or metastatic prostate cancer commonly receive long-term treatment with luteinizing hormone-releasing hormone (LHRH) agonist therapy. This prolonged treatment causes a hypogonadal state of chronic testosterone deficiency. Similar to estrogen deficiency in postmenopausal women, testosterone deficiency among these men negatively affects bone metabolism through a complex self-regulating, negative feedback system and subsequent reduction in bone formation. If left undetected or untreated, the risk for osteoporosis rises. Osteoporosis increases the likelihood of fracture, especially of the hips. Researchers are studying the effects of LHRH agonist therapy on osteoporosis and other related conditions to determine whether interventions, such as pharmacologic agents (e.g., bisphosphonates), dietary supplements (e.g., calcium, vitamin D), and exercise, can slow or prevent the process and assist healthcare providers in knowing how to counsel patients. Current recommendations are found in the literature on glucocorticoid-induced and menopausal osteoporosis. Nurses need to stay abreast of current knowledge in this area, as it is expanding rapidly.

Osteoporosis

Osteoporosis is the most common bone disorder in the United States today, affecting about 15–20 million individuals (Mundy, 1995). A metabolic disorder characterized by decreased bone mass and mechanical support of the skeleton, osteoporosis occurs when the rate of bone resorption greatly exceeds the rate of bone formation. The primary bones affected are the hips, pelvis, wrists, and vertebral column. Although osteoporosis occurs more commonly in postmenopausal women, more than two million men have been diagnosed with the disease (National Osteoporosis Foundation, 1995). Approximately 18 million adults have decreased levels of bone mass, which places them at risk of developing osteoporosis (Lindsay, 2001). The incidence of osteoporosis increases significantly with aging, particularly among men and women over the age of 70 (Zilkoski & Morrow, 1987). Osteoporosis is a major risk factor in fractures (National Institutes of Health [NIH], 2000). When it results in fractures,

Submitted September 2001. Accepted for publication October 30, 2001. This manuscript was prepared as part of the 2001 CJON Mentor/Fellow Writing Program. Ruth Gholz, MS, RN, AOCN®, was paired with Dana Rutledge, PhD, RN, who worked with Francisco Conde, PhD(c), RN, at a National Cancer Institute/Oncology Nursing Society Research Short Course in 1999.

Digital Object Identifier: 10.1188/02.CJON.88-93
Osteoporosis can cause pain, diminished quality of life, decreased physical mobility, inability to work, and increased burden on caregivers of patients with osteoporosis. Treatment of osteoporotic fractures leads to direct financial expenditures of $10–$15 billion annually in the United States (NIH).

**Physiology of the Normal Bone Remodeling Process**

Bone is a living tissue that undergoes a continuous process of remodeling throughout life to maintain its structural and mineral integrity (Watts, 1988). Stable bone mass is dependent on a balanced interaction between osteoblasts and osteoclasts. Osteoclasts are cells that are responsible for forming new bone, whereas osteoclasts are cells that break down or resorb bone. Remodeling throughout the skeleton occurs in discrete packets called the bone multicellular unit or bone-remodeling unit (Frost, 1991; Mundy, 1995). The remodeling process is a coupling of formation to resorption. Histologically, bone formation follows resorption, and this sequence always is the same.

Bone remodeling begins with osteoclast activation. Osteoclasts adhere to bone and remove it. The exact mechanisms by which osteoclasts are activated remain unclear, but the parathyroid hormone, 1,25-dehydroxy vitamin D, interleukin-1, tumor necrosis factor alpha, and growth hormones do stimulate osteoclastic activity. Factors that inhibit osteoclastic activity include calcitonin, estrogen, interleukin-4, interleukin-13, insulin growth factor, transforming growth factor-beta, interferon-gamma, and prostaglandin E (Fleisch, 1995; Mundy, 1995; Rodan, Raisz, & Bilezikian, 1996). In adults without osteoporosis, approximately 2%–10% of skeletal mass undergoes resorption per year (Fleisch).

Following resorption, bone formation occurs, in which resorption sites are refilled by osteoblasts (Fleisch, 1995). Osteoblasts produce the organic matrix of the bone and are found on the outer surface of and inside bones. This matrix gives bones the strength necessary to resist pulling apart (Lindsay, 2001). Weight bearing and exercise independently stimulate bone formation (Mundy, 1995). Osteoblastic bone formation is estimated to take about three to four months to replace the bone that has been resorbed by osteoclastic activity in less than two weeks (Mundy; Rodan et al., 1996). Normally, the amount of bone formed equals the amount of bone destroyed so that no change in the shape of the bone occurs.

### Osteoporosis in Men

Osteoporosis and reduction of bone mass occurs when an imbalance exists between osteoclastic bone resorption and osteoblastic formation. Although a great deal of research has focused on postmenopausal osteoporosis, relatively little has been studied about osteoporosis in men. Androgens and testosterone play a vital role in bone metabolism, and deficiency in these hormones reduces osteoblastic formation. Testosterone deficiency is analogous to estrogen deficiency in menopausal women as it relates to bone mineral density (BMD). These factors may be responsible for the bone loss evident in men with primary or secondary hypogonadism (Ybarra, Ade, & Romeo, 1996).

In vitro studies have shown the presence of androgen receptors in osteoblasts (Colvard et al., 1989; Orwell, Stribiska, Ramsey, & Keenan, 1991). Androgens can directly stimulate bone cells to proliferate and also can inhibit bone resorption (Kasperk et al., 1988; Vanderschueren & Bouillon, 1995). In animal models, total bone mass was decreased in both young and adult androgen-deficient rats (see Table 1) (Vanderschueren & Bouillon).

When androgen replacement was administered in rats, cortical thinning of the femoral shaft associated with the normal aging process was prevented, indicating that decreased serum testosterone in older male rats stimulated resorption of endosteal bone (Vanderschueren et al., 1993). These preclinical studies suggest that androgens may have both inhibitory effects on bone resorption and stimulatory effects on periosteal bone formation. Therefore, in the absence of androgens, the lack of osteoblastic bone formation coupled with an increased rate of osteoclastic activity may cause reduction in bone mass and eventually lead to osteoporosis.

### Luteinizing Hormone-Releasing Hormone Agonist Therapy

The term “hypothalamic-pituitary-gonadal axis” often is used to describe the reproductive hormonal system in men. This negative feedback system is self-regulating, consisting of the hypothalamus, pituitary gland, and testes (Small & Prins, 1996). The prostate gland consists of stromal and epithelial cells. Protein synthesis and growth of these prostate cells are largely dependent on the presence of male hormones, specifically, androgens (see Figure 1). The hypothalamus produces LH, or gonadotropins, and cortisol-releasing factor. Endogenous LHRR stimulates the receptors in the anterior pituitary to produce the luteinizing hormone (LH), follicle-stimulating hormone (FSH), prolactin, and adrenocorticotropic hormone (ACTH) (Small & Prins, 1996). LH stimulates the Leydig cells of the testicles to produce testosterone, whereas ACTH stimulates the adrenal gland to produce androgens that subsequently are converted into testosterone. About 95% of total testosterone comes from the testes in adult men, with the adrenals secreting the remaining 5% (Plosker & Brogden, 1994). Testosterone and estrogen regulate the pituitary release of LH and FSH via a negative feedback mechanism, and this determines LHRR release from the pituitary (Plosker & Brogden). Testosterone then passively enters prostate cells where it is converted by the enzyme 5-alpha-reductase into dihydrotestosterone (DHT). DHT is the active metabolite that influences the growth and protein synthesis of prostate cells (McLeod & O’Brien, 1996).

The signals from the secretion of endogenous LHRR are essential for the maintenance of the hypothalamic-pituitary-gonadal axis. Continued exposure from the administration of exogenous LHRR (such as that given in prostate cancer) ablates the normal pattern, resulting in pituitary desensitization and subsequent inhibition of LH and FSH production (Small & Prins, 1996). Initial administration of LHRR agonists causes a sharp rise in LH and FSH levels, leading to increased testicular androgen production. The rise in androgen levels is transitory, usually lasting seven days or less. However, during this transient rise in androgen levels, patients may complain of symptom “flares” that may be reduced by coadministration of an antiandrogen agent one week prior and up to four weeks after the initial administration of an LHRR agent (Plosker & Brogden, 1994). Regardless of the type of LHRR agonist used, continuous administration causes

### Table 1. Skeletal Changes in Androgen-Deficient Rats

<table>
<thead>
<tr>
<th>Skeletal Changes</th>
<th>Young Rats</th>
<th>Old Rats</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body weight</td>
<td>Decreased</td>
<td>Unchanged</td>
</tr>
<tr>
<td>Bone growth and modeling</td>
<td>Decreased</td>
<td>Unchanged</td>
</tr>
<tr>
<td>Total bone mass</td>
<td>Unchanged</td>
<td>Decreased</td>
</tr>
<tr>
<td>Cortical bone density</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Cancellous bone density</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
</tbody>
</table>

Note. Based on information from Vanderschueren & Bouillon, 1995.
a significant and sharp decline in testicular androgen level to castration level (McLeod & O’Brien, 1996; Plosker & Brogden). Hypogonadism (defined as serum testosterone less than 10 nmol/L) usually is achieved within three to four weeks and can be maintained for the duration of treatment (Plosker & Brogden). Up to 80% of patients with local or metastatic prostate cancer are estimated to partially or completely respond to androgen suppression (Wolciechowski et al., 1986).

Several studies document the effects of LHRH agonist therapy on BMD of patients with prostate cancer. World Health Organization standards for interpreting bone density measurements are found in Table 2. In nine French men treated with LHRH agonist therapy for stage C prostate cancer, Maillefer et al. (1999) found that BMD decreased, particularly in the femoral head. Serum testosterone and LH levels were significantly lower at months 6, 12, and 18 as compared to month 0. In a retrospective study, Townsend, Sanders, Northway, and Graham (1997) determined the incidence of bone fractures in men with various stages of prostate cancer who received LHRH agonist therapy between 1988 and 1995. Overall incidence of osteoporotic fractures in that study was 5% (11 of 224); this is triple the incidence of fractures in normal men of similar ages. However, 9 of 11 osteoporotic fractures that occurred were seen predominantly among patients with stage D or metastatic disease.

In two studies (Revilla et al., 1998; Rico et al., 1996), investigators examined total body bone mineral content and total body BMD using a dual x-ray energy absorptiometry (DEXA) scan in men with stage D, metastatic prostate cancer and in age-matched controls. Patients with prostate cancer received total androgen blockade therapy. These studies suggest that bone destruction in patients with bone metastases may result from an increased osteolytic, rather than osteoblastic, feature of the metastases.

Diamond, Campbell, Bryant, and Lynch (1998) followed 12 men with metastatic prostate cancer who were treated with combined androgen suppression therapy. After six months of treatment, patients received adjuvant intermittent etidronate therapy and a calcium supplement for 6 or 12 months. Serum and urinary markers of bone turnover also increased significantly in healthy controls over one year. Markers of bone turnover also increased significantly during this initial six-month period. With the administration of etidronate and calcium therapy, mean spinal QCT increased by 8%. This suggests that pharmacologic agents may help reverse the trend toward bone loss.

Other Factors That May Cause Bone Loss in Men

A myriad of factors have been reported to contribute to low BMD and increase the risk of osteoporosis in men. These include aging, ethnicity, diet, stress, physical inactivity, low body mass index, certain medications, and disease.

The average adult develops peak bone mass by the age of 30. The reduction in this bone mass is affected by genetic, nutritional, hormonal, and environmental factors. Generally, women peak in bone mass during adolescence and almost have completed bone mass formation when puberty ends. For men, puberty starts later and lasts longer, thus allowing for a greater bone mass and skeletal structure as compared to women. Trabecular bone is crucial in assessing osteoporosis and risk factors. With normal aging, both men and women lose trabecular bone at the spine and iliac crest, yet the pattern of bone loss differs. Women have trabecular perforation or microfractures of the trabeculae, placing them at greater risk of fracture. Trabecular perforation is not noted in aging men, which may lead to better responses to bone-forming therapies (Siddiqui, Shetty, & Duthie, 1999).

In healthy men, the mean testosterone level declines with age. Low testosterone levels predict lower BMD, especially in the femoral neck (Kenny, Prestwood, Marcello, & Raisz, 2000). Thus, older men with low

![Hypothalamic-Pituitary-Gonadal Axis System](image)

**Note.** Based on information from Small & Prins, 1996.

<table>
<thead>
<tr>
<th>Stage</th>
<th>Bone Mineral Density Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Bone mineral density (BMD) value not more than 1 standard deviation (SD) below the young adult mean value</td>
</tr>
<tr>
<td>Osteopenia</td>
<td>BMD value between 1 and 2.5 SD below the young adult mean value</td>
</tr>
<tr>
<td>Osteoporosis</td>
<td>BMD value more than 2.5 SD below the young adult mean value</td>
</tr>
<tr>
<td>Established osteoporosis</td>
<td>BMD value more than 2.5 SD below the young adult mean value in the presence of one or more fractures</td>
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**Note.** Based on information from Blake & Fogelman, 1997.
testosterone levels are more likely to be at risk for hip fracture.

Although black men usually have a higher BMD than their white counterparts (Barondon, Nelson, & Schlaen, 1997), they have a higher fracture rate (Anderson & Pollitzer, 1994). The cause of this higher fracture rate is unknown.

Several studies (Daniell, 1997; Daniell et al., 2000; Eriksson, Eriksson, Stege, & Carlstrom, 1995; McGrath & Diamond, 1995) have examined the effects of therapeutic orchiectomy on BMD in patients with prostate cancer. These studies suggest an association between BMD loss in patients who have had orchiectomy and an increased incidence of osteoporotic fractures.

Medical Management of Osteoporosis

The literature is saturated with information about pharmacologic management of osteoporosis in women; yet it is scant for men (Daniell, 2001). Information about managing osteoporosis comes from the available literature, which means some treatments are untested in men. Bisphosphonates have proven effective in the prevention and treatment of osteoporosis and osteopenia (Woo & Adachi, 2001). These agents inhibit osteoclast activity, thus reducing bone resorption and allowing a gain in bone mass. Clinically, bisphosphonates lead to reduction in vertebral and nonvertebral fractures.

At present, both IV and oral bisphosphonates exist (Mycek, Harvey, & Champe, 2000). Etidronate, risedronate, and alendronate are taken orally. Because oral absorption of these agents is less than 10%, they must be administered on an empty stomach with at least six to eight ounces of water. Although alendronate is the only bisphosphonate approved by the U.S. Food and Drug Administration for men with osteoporosis, IV pamidronate has been shown to prevent bone loss in men with prostate cancer who are receiving a gonadotropin-releasing hormone agonist (Smith et al., 2001). Pamidronate is an IV bisphosphonate available for patients who cannot tolerate the oral route.

For all of these drugs, diarrhea, nausea, abdominal pain, and occasional incidences of esophageal ulcers are side effects that require special consideration (Mycek et al., 2000). All patients receiving bisphosphonates must be monitored for adequate calcium and vitamin D intake, as the use of bisphosphonates generally has been tested in the context of supplemental calcium and vitamin D (Heaney, 2001). Another pharmacological agent that may be used to treat osteoporosis is calcitonin, a supplement given nasally that increases osteoclastic activity (Daniell, 2001; Woo & Adachi, 2001).

Implications for Practice

Widespread use of serum prostate-specific (PSA) for prostate cancer screening has brought significant improvements in early detection, along with new challenges. Because widespread PSA screening began in 1988, prostate cancer incidence rose abruptly and peaked in 1992. Since 1992, incidence has declined steadily, and more men are being diagnosed with local and regional disease. Clinicians now see a growing population of men with PSA recurrence or biochemical relapse following treatment with radical prostatectomy or radiation therapy. Once used primarily as palliative therapy for men with distant metastasis, the use of LHRH agonists has expanded to include those with biochemical relapse. Although when to initiate LHRH agonist therapy remains controversial, administration is lifelong. Prolonged treatment causes a hypogonadal state in these men that may negatively affect bone metabolism and,

Case Study

C.J. is a 64-year-old male diagnosed with advanced prostate cancer in 1992. At the time of diagnosis, his prostate-specific antigen (PSA) was 159 and his Gleason score was 7. He had positive peritoneal and iliac nodes, as well as bone metastases in his ribs, pelvis, spine, and skull. His minimal bone pain was resolved with ibuprofen every eight hours and oxycodone/acetaminophen as needed (he averaged one per day).

C.J.’s treatment consisted of a combination of leuprolide and flutamide that brought his PSA to 0.5 and led to complete pain relief. He was fully functional and worked full-time for six years while on this regimen. In 1998, he began to complain of increased pain in his pelvis and lower back. His PSA remained at 0.7, and a bone scan indicated osteoarthritis of the thoracic spine and feet. He began a course of sustained release morphine twice daily with relief.

When examined in 2000, C.J.’s PSA had risen to 7.0. He received radiation to the prostate, and his pain increased despite no evidence of bone metastasis.

In 2001, C.J.’s PSA was 21. His pain had progressed to include both hips, femurs, and his entire spine, and he required twice daily 30 mg sustained release morphine.

After referral for bone density examination, C.J. was diagnosed with severe osteoporosis as evidenced by bone density studies of the lumbar spine and proximal right femur. The report follows.

C.J. was placed on calcium and vitamin D supplementation and began an exercise program developed specifically for him from a physical therapist. Bone density examinations will be repeated every six months with treatment adjusted accordingly.

Report

A bone density study of the lumbar spine and proximal right femur was acquired and analyzed using a dual energy x-ray absorptiometry bone densitometer.

Summary findings for bone mineral density (BMD) follow.

- L2–L4 BMD (g/cm²) 0.933 +/- 0.01
- L2–L4 % young adult 75 +/- 3, T = -2.56
- L2–L4 % age-matched 77 +/- 3, Z = 2.30

Results for individual vertebrae: L1 0.770 g/cm² (T = -3.25), L2 0.822 (T = -3.48), L3 0.983 (T = -2.15), and L4 0.990 (T = -2.08).

- Femoral neck BMD (g/cm²), 0.709 +/- 0.01
- Femoral neck % young adult, 66 +/- 3, T = -2.78
- Femoral neck % age-matched, 72 +/- 3, Z = 2.14

T and Z scores are the number of standard deviations (SD) that the patient’s BMD is above (+) or below (–) the mean value for the reference population (i.e., young adults or age-matched). The T score is the most accurate for the diagnosis of osteoporosis, prediction of fracture, and response to therapy. The Z score is an age-matched comparison, adjusted for patient’s weight and ethnicity and is of lesser clinical utility. A T score of +/- 1 SD is normal. According to the World Health Organization standards (Blake & Fogelman, 1997), a T score of –1 to –2.5 is indicative of osteopenia and a T score greater than –2.5 is indicative of osteoporosis.

Impression: Osteoporosis of the lumbar spine and right hip.

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if left undetected, could increase the risk for osteoporosis.

The prevalence of prostate cancer and the economic cost of its treatment are expected to increase as the male aging population continues to grow and more men are diagnosed with early disease resulting in longer survival. However, the quality of this survival may be affected adversely by osteoporosis. If patients at risk can be identified, targeted interventions can be instituted. Nursing interventions (e.g., calcium supplements, exercise) may be needed that will not affect the antitumorogenic effects of LHRH agonist therapy but decrease the risk of osteoporosis.

Nurses are challenged on a daily basis to “stay alert” while assessing patients and preventing potential problems. Osteoporosis is a newly recognized complication of androgen depletion. Healthcare professionals should educate male patients about the risks of androgen depletion and strategies that may reduce the long-term sequelae.

Evidence-based treatment and prevention of osteoporosis is in its infancy. Although studies examining the utility of agents such as calcitrol and etidronate are ongoing (Cortet et al., 2001; Summers, 2001), men can be counseled using guidelines for prevention and treatment of osteoporosis for other causes (e.g., glucocorticoid-induced, menopausal) (American College of Rheumatology Ad Hoc Committee on Glucocorticoid-Induced Osteoporosis, 2001; Elliott, Farrah, Binkley, Carnes, & Gudmundsson, 2000; Goodman, 1996). These guidelines have been developed based on the physiology of bone growth and breakdown. Following these recommendations may or may not prevent osteoporosis in men with prostate cancer undergoing hormone ablation; however, none of the recommendations is harmful.

Intake of calcium and vitamin D are critical to maintaining normal bone formation, and normal aging reduces calcium absorption. Men should be encouraged to have a high dietary intake of dairy food and cereals with added calcium. Supplements may be needed to meet minimum daily requirements (none have been specified for men with prostate cancer). Calcium carbonate has more absorbable calcium than other forms. Calcium supplements taken with food, especially acidic foods (e.g., citrus juices), are absorbed better. Vitamin D normally is adequate with some sun exposure. Multivitamins often contain 400 international units of vitamin D, which is a “supplemental” amount. Each patient’s dietary habits require review to optimize calcium and vitamin D intake or supplementation.

Certain exercise may help bones maintain a solid matrix. This includes weight-bearing exercise that puts stress on bones, such as stair climbing, racket sports, jogging, and weight training. The exercises usually preferred by older men—walking, bicycling, and swimming—do not increase bone strength. Men who normally walk, bicycle, or swim should be encouraged to supplement these activities with realistic weight-bearing exercise (e.g., lifting milk cartons filled with water, pulling against bungee cords attached to a door). Exercise performed for 30–60 minutes three to four times per week is necessary to strengthen bone and increase muscle mass and strength. When possible, older men can be referred to facilities that have exercise programs geared to “seniors” or people with chronic diseases.

Smoking has deleterious effects on bone strength and mass (Lin, Chen, Chang, & Ho, 2001; Orwell, Bevan, & Phipps, 2000; Vogel, Davis, Nomum, Wasnich, & Ross, 1997). Smoking cessation programs can be sought as appropriate.

If men with osteoporotic bones fall, they are at risk for fracture. Neurologic disease, older age, balance disorders, and comorbidity place patients at higher risk. Alcohol abuse has been associated with osteoporotic hip fractures secondary to falls (Lau et al., 2001; Summers, 2001). Oncology nurses must continue to support the mission of quality cancer care for men with prostate cancer and bring the issue of osteoporosis to the forefront. As is commonly known, “an ounce of prevention is better than a pound of cure.”

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References
Lau, E.M., Suriwongpaisal, P., Lee, J.K., Das,
Evidence-based treatment and prevention of osteoporosis for men is early in its science as compared to its treatment and prevention in postmenopausal women. Osteoporosis can negatively affect quality of life by leading to pain and depression, and fractures can affect mobility, finances, and caregiver burden.

Androgens and testosterone play a vital role in bone metabolism. In the absence of androgens, the lack of osteoblastic bone formation coupled with an increased rate of osteoclastic activity causes reductions in bone mass that eventually lead to osteoporosis.

Luteinizing hormone-releasing hormone (LHRH) agonist agents are the mainstay of therapy in advanced prostate cancer. A sustained hypogonadal state from chronic use of LHRH agonist agents may place men at risk for osteoporosis. Men with prostate cancer require ongoing surveillance for bone strength and grading.

Osteoporosis can negatively affect quality of life by leading to pain and depression, and fractures can affect mobility, finances, and caregiver burden.

Evidence-based treatment and prevention of osteoporosis for men is early in its science as compared to its treatment and prevention in postmenopausal women.

Oncology nurses play a key role in educating these men regarding the potential adverse effects of osteoporosis from LHRH agonist therapy and in advocating for the appropriate assessment and preventative measures.